



Research Article

Early Stage Hip Osteoarthritis is Associated with Specific Muscle Weakness

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Abstract

Study background

Knowledge regarding muscle function (e.g. muscle strength and power) in hip osteoarthritis and especially which muscles that are most affected is scarce and muscle function in the earlier stage of hip osteoarthritis is especially unexplored.

Methods

In an explorative cross-sectional study we examined deficits in muscle function in 60+ year old patients from a randomized study that investigated the effects of exercise in clinically verified unilateral hip osteoarthritis in the absence of x-ray verified joint damage (n=46). A paired t-test was used to examine whether there was a significant deficit in muscle function between the symptomatic and the non-symptomatic lower extremity and the magnitude of the side difference was presented as a %-difference. Muscle function was assessed as maximal isometric hip and thigh muscle strength and leg extensor power.

Results: These patients displayed significant deficits in hip flexor (difference mean \pm SD (95% CI)= -3.5 \pm 9.2 Nm (-6.2, -0.8); %-difference: 7.6%); and abductor strength (-3.6 \pm 10.4 Nm (-6.7, -0.5); 6.3%), knee extensor strength (-9.1 \pm 13.6 Nm (13.2, -5.1); 8.0%) and leg extensor power (-10.0 \pm 22.8 W (-16.8, -3.2); 8.1%).

Conclusion: Our results suggest that in the early stage of hip osteoarthritis there may be a pattern of localized weakening of some of the hip and thigh muscles.

Keywords

Hip osteoarthritis; Muscle strength; Muscle weakness; Muscle strength dynamometer; Outcome measures

Abbreviation: ACR: American College of Rheumatology; CV: coefficient of variation; HOOS: the hip disability and osteoarthritis outcome score; ICC: intraclass correlation coefficient; MRI: magnetic resonance imaging; Nm: Newton meter; OA: osteoarthritis; RCT: randomized controlled trial; SD: standard deviation; W: Watt; WOMAC: the Western Ontario and McMaster Universities Osteoarthritis Index LK 3.0.

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Introduction

Hip and knee osteoarthritis (OA) is one of the leading causes of disability globally and due to the increasing prevalence of obesity around the world and the demographic changes health profession need to prepare for a large increase in the demand for health services to treat hip and knee OA [1]. Thus, early treatment to prevent or slow down the disease process is warranted. A more thorough understanding of the musculoskeletal factors underlying functional limitations in OA could be a key element for achieving this goal. Increasing evidence suggests that in knee OA muscle strength, specifically knee extensor muscle weakness, is associated with increased risk of developing knee OA [2] and increased risk of symptomatic and functional deterioration [3]. The role of knee extensor muscle weakness as a risk factor for development of knee OA is not fully understood but the knee extensors work as shock absorbers and stabilizers, and hence protect the joint surface during loading and movement [2]. In knee OA strengthening exercise is an essential part of the recommendations for the non-pharmacological core management [4] and specifically knee extensor exercises seem to be more beneficial for reducing pain and disability than more general exercises for the lower extremities [5].

Whether low muscle strength is a risk factor for development of hip OA is unknown, but compared to healthy older adults patients with hip OA have reduced hip and thigh muscle strength [6]. One longitudinal study suggested that reduced hip abductor muscle strength is associated with more limitations in activities in hip OA [7]. However, the literature on muscle function (e.g. muscle strength and power) and especially which muscles that are most affected in patients with hip OA is scarce, and exercise prescriptions in hip OA are largely based on expert opinion [6]. A systematic review concluded that hip OA may result in generalized muscle weakening of the affected lower extremity rather than a localized weakening of the hip muscles per se, but this conclusion was primarily based on studies of patients with end-stage hip OA awaiting hip replacement [6]. We have recently demonstrated that the global muscle weakness of the affected lower extremity also applies for patients with symptomatic and radiographically verified unilateral hip OA who are not on waiting list for hip replacement [8]. These patients had a significantly lower hip and thigh muscle strength of the symptomatic lower extremity compared to the non-symptomatic lower extremity and the largest numerical side difference in muscle function was evident for leg extensor power [8]. Very little is known about muscle function in the earlier stage of hip OA

Radiography has been the main method used to define osteoarthritis but when the OA diagnose is confirmed radiographically, there is already significant joint damage [9]. The use of MRI and arthroscopy have made it clear that early osteoarthritic changes are not apparent on the radiograph [9].

Thus the aim of this cross-sectional study was to explore if maximal isometric hip and thigh muscle strength, and leg extensor muscle power of the symptomatic lower extremity were significantly lower compared to the non-symptomatic lower extremity in 60+ year old patients who met the clinical criteria for unilateral hip OA but

where joint damage could not yet be verified by traditional x-ray. Since the study was explorative we had no pre-specified hypothesis.

Patients and Methods

Patients

In this exploratory, cross-sectional study we included a subsample of patients with unilateral, symptomatic hip OA who met the clinical classification criteria of hip OA according to American College of Rheumatology (ACR) criteria regarding classification of hip OA [10], but not the radiographic criteria for hip OA according to the Kellgren-Lawrence grading [11] based on a supine anterior-posterior x-ray. The main study was a randomized controlled trial (RCT) (ClinicalTrials.gov identifier: NCT01387867) that investigated the effects of exercise in 60+ years old patients with hip OA [12]. Inclusion criteria for the RCT were home-dwelling 60+-year old individuals with clinical hip OA according to the ACR criteria [10], who were not on a waiting list for hip replacement. Exclusion criteria were 1) symptomatic OA of the knee or the big toe or 2) other types of arthritis, 3) previous hip or knee replacement or 4) hip fracture, 5) co-morbidity that prevented exercising, 6) treatment related to hip problems within the last 3 months or 7) performing regular exercise/sports twice or more weekly and 8) inability to use public transportation [12]. The patients were recruited through general practitioners or specialists and advertisements in local newspaper. The experimental protocol described in detail elsewhere [12] was in compliance with the Helsinki Declaration and the Danish Ethics Committee of the Capital Region approved the study (H-C2009-42). Signed informed consent was obtained from all participants.

Methods

Muscle function assessments: A detailed description of the maximal isometric muscle strength and leg extensor power measurements has been published previously [8,13]. Briefly, all measurements were conducted by two experienced physiotherapist using the exact same standardized test conditions and protocol [8,13]. The non-symptomatic lower extremity was tested first and measurements were repeated until a decrease in output occurred. The highest value was used for data analysis. A minimum of three repetitions for the strength measurement and five repetitions for the leg extensor power measurement was required.

Hip muscle strength was measured with a handheld dynamometer (JTech Power Track II) commander [8,13]. Hip abductor and adductor strength was measured with the patients in supine position while external and internal rotator and flexor strength was measured with the patients seated in a straight-back chair with hips and knees flexed at 90 degrees.

Thigh muscle strength was measured with the Good Strength device (Version 3.14 Bluetooth; Metitur Ltd., Finland) and the patients were seated with hips flexed to 90 degrees and knees flexed to 60 degrees [8,13,14].

The lever arm for the muscle strength measurements was determined as the measured distance from the transducer or force pad to the joint axis of rotation [8,13].

Maximum single leg extensor power (force x velocity) was measured using a Leg Extensor Power Rig (Queen's Medical Centre, Nottingham University, UK) [8,13,15]. The patients were in a seated position and a single explosive lower limb extension accelerated a

flywheel from rest and the maximum speed of the flywheel was used to calculate the average power of the lower limb extensor muscles [15].

Descriptive variables: The patients self-reported pain, stiffness and function were assessed using the Western Ontario and McMaster Universities Osteoarthritis Index LK 3.0 (WOMAC) [16] which is included in its complete and original format in The hip disability and osteoarthritis outcome score (HOOS) questionnaire [17]. The WOMAC Index consists of 3 subscales scores: pain (0-20 (best to worst)), stiffness (0-8) and function (0-68).

Statistical analysis: Normally distributed data are presented as mean \pm standard deviation (SD) and non-normally distributed data as median and interquartile range (25-75 percentiles). A paired t-test was used to examine whether there were significant differences in hip and thigh muscle strength and leg extensor power between the symptomatic and the non-symptomatic lower extremity and the difference in values are presented as mean \pm SD and the 95% confidence interval. A significant difference in muscle function between the two legs was defined as deficit or side difference and the magnitude of side difference presented as a ratio: (the symptomatic extremity/non-symptomatic lower extremity) x 100 and a %-difference: ((the non-symptomatic lower extremity - the symptomatic lower extremity)/the non-symptomatic lower extremity) x 100. Data were analyzed using SPSS, version 20 (SPSS Software Inc., Chicago, IL).

Results

Forty-six patients with relatively mild symptoms from unilateral early stage hip OA were included in the study. The patients mean age was 70 years. Other characteristics are shown in Table 1. Hip flexor and abductor strength, knee extensor strength and leg extensor power were significantly lower in the symptomatic lower extremity compared to the non-symptomatic lower extremity (Table 2). The deficits ranged from a %-difference of 6.3% in hip abductor strength to 8.1% in leg extensor power. There were no deficits in strength of the hip adductors, external or internal rotators or knee flexors (Table 2).

Discussion

In patients with early stage hip OA who met the clinical but not the radiographic criteria for unilateral hip OA, hip flexor and abductor strength, knee extensor strength and leg extensor power of the symptomatic lower extremity were significantly lower than the non-symptomatic lower extremity.

We could only identify one study that has investigated deficits in muscle function in patients with unilateral hip OA based on the clinical ACR-classification criteria that did not include patients on waiting list for hip replacement [18]. However, this study [18] did not

Table 1: Patient characteristics.

Sex (female/male)	25/21
Age (years)	70 \pm 7
Height (cm)	169 \pm 8
Weight (kg)	79 \pm 15
BMI (kg/m ²)	27 \pm 4
WOMAC Pain (0-20)	5 (3-8)
WOMAC Stiffness (0-8)	3 (2-4)
WOMAC Function (0-68)	14 (8-22)

Note: Values are mean \pm SD, median (interquartile range, 25-75 percentiles) or number; BMI = Body mass index; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index

Table 2: Deficits in muscle function between the symptomatic and the non-symptomatic lower extremity (n=46).

Strength & power	Symptomatic/	Difference (95% CI)	Ratio (%) %-difference
	Non-symptomatic		
Hip (Nm)			
XExternal rotators	31.2 ± 13.4/	-0.5 ± 4.8	98.2 ± 17.6/
	31.7 ± 12.5	(-1.9-0.9)	1.8 ± 17.6
XInternal rotators	31.0 ± 13.1/	-0.4 ± 6.1	98.5 ± 19.8/
	31.4 ± 11.8	(-2.2-1.4)	1.5 ± 19.8
XFlexors	43.6 ± 20.0/	-3.5 ± 9.2*	92.4 ± 18.7
	47.1 ± 18.6	(-6.2- -0.8)	7.6 ± 18.7
XAbductors	50.0 ± 23.3/	-3.6 ± 10.4*	93.7 ± 20.1/
	53.6 ± 23.5	(-6.7- -0.5)	6.3 ± 20.1
XAdductors	57.3 ± 27.2/	-0.1 ± 11.9	99.4 ± 18.9/
	57.2 ± 24.8	(-3.5-3.6)	0.6 ± 18.9
Thigh (Nm)			
XExtensors	105.8 ± 46.0/	-9.1 ± 13.6**	92.0 ± 12.5/
	114.9 ± 46.9	(-13.2- -5.1)	8.0 ± 12.5
XFlexors	52.6 ± 27.4/	0.4 ± 7.8	102.0 ± 19.7/
	52.3 ± 27.4	(-2.0-2.7)	-2.0 ± 19.7
Power (Watt)			
XLeg extensor power	124.2 ± 62.0/	-10.0 ± 22.8**	91.9 ± 16.2/
	134.2 ± 61.8	(-16.8- -3.2)	8.1 ± 16.2

Note: Values are mean ± SD or as indicated. Significant difference between the symptomatic and the non-symptomatic lower extremity; * p<0.05, **p<0.01. 95% CI = 95% confidence interval; Nm = Newton meter.

report any information regarding joint damage or findings on x-ray. Thus we only know that these patients had relatively mild symptoms of OA and that they signed up for an RCT regarding the effect of exercise in patients with hip or knee OA. In line with our previous results in patients with clinical and radiographically verified (Kellgren-Lawrence ≥ 2) unilateral hip OA with relatively mild symptoms [8]. This study [18] showed a generalized weakening of the symptomatic lower extremity compared to the non-symptomatic lower extremity. In contrast, our patients in the present study only had significant deficits or side differences in hip flexor and abductor strength, knee extensor strength and leg extensor power. These results suggest that in the early stage of hip OA there may be a pattern of localized weakening of some of the hip and thigh muscles. Furthermore, the patients with early stage of hip OA had scores for WOMAC pain subscale similar (p=0.625) to the patients with clinical and radiographically verified unilateral hip OA [8], but better scores for WOMAC subscale stiffness (p=0.015) and physical function (p=0.002).

The significant deficit in hip flexor strength may be an early indication of the initial phases of OA. Inflammation of the synovial membrane occurs in both early and late phases of OA and is directly linked to clinical symptoms such as joint swelling, synovitis and inflammatory pain [19]. These clinical symptoms may potentially affect the force production of the most important hip flexor the psoas muscle because it passes the hip joint only separated from the joint capsule by bursa iliopectinea, which can communicate with the hip joint cavity [20].

Although abnormal function of the key hip stabilizer muscles such as the hip abductors have been linked to hip OA, studies investigating the structure and function of the hip abductors in populations with OA have reported conflicting results [21]. A systematic review from 2013 regarding muscle weakness in hip OA concluded that deficits in hip adductor and abductor strength were less consistent [6]. In contrast, a systematic review from 2016 regarding structure and function of the hip abductors in hip OA found that the unaffected side was stronger

than the affected side [21]. However, nearly all the studies included in both systematic reviews primarily recruited patients with end-stage hip OA. We found only one study [22] that has investigated deficit in hip abductor strength in patients with hip OA based on the clinical ACR-classification criteria. While our results suggest that even in the early stage of hip OA deficit or side difference in hip abductor strength is present that study [22] showed no side difference in hip abductor and adductor strength. However the authors did not provide any information regarding joint damage or findings on x-ray, and the study included patients with bilateral hip OA and patients awaiting hip replacement, which may have influenced the results.

In the early stage of hip OA, deficits in hip flexor and abductor strength may be a consequence of gait alterations due to hip pain. Pelvic and lower extremity compensatory action in the gait such as increase in the pelvic obliquity on the weight-bearing limb has been demonstrated in patients with earlier stage hip OA (Kellgren-Lawrence score < 3) [23]. The increased pelvic obliquity minimizes the load on the painful hip corresponding to a Trendelenburg lurch, which reduces the load of the hip by decreasing the hip abductor activity [23]. Although, the initial underlying cause of the adaptation is pain and not muscle weakness, muscle weakness may develop secondarily from continually walking with a decreased demand on certain muscle group [24]. Muscle-driven gait simulations have shown that gait appears most robust to weakness of hip and knee extensors and most sensitive to weakness of e.g. hip abductors and flexors [25], suggesting that focusing on strengthening these muscle groups may be important for effective rehabilitation in early stage hip OA.

We could not find any study that has investigated deficit in knee extensor strength or leg extensor power in hip OA based on the clinical ACR-classification criteria, except our own study, but these patients had radiographically verified unilateral hip OA [8]. Like these patients, the patients with early stage, unilateral hip OA have a side difference in knee extensor strength and leg extensor power. We have previously shown that healthy peers, when measured with the

same device and method, have no side difference in the leg extensor power [8]. In addition, a population-based study has shown that older adults with hip pain have low leg extensor power in comparison to peers without hip pain, and hip pain is not associated with e.g. muscle strength measures of hip abductors/adductors or flexors/extensors or knee extensors/flexors [26]. These results could indicate that leg extensor power may be an appropriate measure to detect early deficits in muscle function in patients with hip OA. The leg extensor power measurement appears to be a reproducible measure in patients with hip OA (test-retest reliability ICC: 0.93-0.96 and agreement CV: 8-10%) [13].

It is a limitation of the study that we did not measure hip extensor strength. However, it is a challenge to test the hip extensor strength with a handheld dynamometer in patients with hip OA because the traditional muscle test requires that the patient is in the prone position [27,28].

Patients with symptomatic hip OA may not tolerate the prone position well and limitations in spine and hip mobility also make it difficult to perform a valid assessment of hip extensor strength [27,28].

Conclusion

To our knowledge, this is the first study that has explored muscle function in the earlier stage of hip OA where the patients meet the clinical criteria for unilateral hip OA but where joint damage cannot yet be verified by traditional x-ray. Our results showed a significant deficit in hip flexor and abductor strength, knee extensor strength and leg extensor power of the symptomatic lower extremity compared to the non-symptomatic lower extremity suggesting that this patients group may benefit from strengthening exercises for these muscle groups. Healthy peers do not show a side difference in leg extensor power [8], and thus assessment of leg extensor power may be an appropriate measure to detect early deficits in muscle function in patients with hip OA. Since the study was explorative replication of the results is needed and longitudinal research is required to determine if the leg extensor power measurement is sensitive to measure change in muscle function over time in patients with early stage hip OA.

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Declaration of conflicting interest

The authors report no conflicts of interest.

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